AD-759 510

QUANTITATIVE INDICIES OF THE SHOCK SYNDROME

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Prepared for:

Army Medical Research and Development Center

April 1973

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FINAL REPORT

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La Jolla, California 92037

April 1973

Supported by

U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
Washington, D. C. 20315

CONTRACT NO. DA DA 17-67-C-7001

Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified

1. ORIGINATING ACTIVITY (Corporate author)
AMES-Bioengineering
University of California, San Diego
La Jolla, California 92037

2a. REPORT SECURITY CLASSIFICATION

2b. GROUP

3. REPORT TITLE
Quantitative Indicies of the Shock Syndrome

4. DESCRIPTIVE NOTES (Type of report and inclusive dates)
Final Report, 1 July 1966 - 30 September 1973

5. AUTHOR(S) (First name, middle initial, last name)
B. W. Zweifach

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
April 1973	2	0

8a. CONTRACT OR GRANT NO	9a. ORIGINATOR'S REPORT NUMBER(S)
DADA17-67-C-7001	
b. PROJECT NO.	
c.	9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report)
d.	

10. DISTRIBUTION STATEMENT
Approved for public release; distribution unlimited

11. SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY
	US Army Medical Research and Development, Command, Washington, DC 20314

13. ABSTRACT

Two forms of experimental shock were investigated - hemorrhagic and bacterial endotoxemia - in the dog, cat and rabbit. In addition to measurement of central arterial pressure, central venous pressure, heart rate, respiratory rate, plasma pO_2, pCO_2 and pH, blood hematocrit, and plasma colloid osmotic pressure, observations were carried out on the microcirculation of the mesentery and omentum.

DD FORM 1 NOV 65 1473 REPLACES DD FORM 1473, 1 JAN 64, WHICH IS OBSOLETE FOR ARMY USE.

Security Classification

	ROLE	WT	ROLE	WT	ROLE	WT
experimental shock						
hemorrhagic shock						
bacterial endotoxemia						
arterial pressure						

Two forms of experimental shock were investigated - hemorrhagic and bacterial endotoxemia - in the dog, cat and rabbit. In addition to measurement of central arterial pressure, central venous pressure, heart rate, respiratory rate, plasma pO_2, pCO_2 and pH, blood hematocrit, and plasma colloid osmotic pressure, observations were carried out on the microcirculation of the mesentery and omentum. At the microvascular level measurements were made of micropressure, flow, fluid exchange and vascular permeability.

(1) It was found that measurements of colloid osmotic pressure of the plasma ($\pi p\ell$) during the progression of circulatory insufficiency was a reliable index of the state of the individual. Compensation was associated with hemodilution and a lowering of $\pi p\ell$ by some 15-20%. This level was maintained for several hours, after which $\pi p\ell$ levels tended to rise. The latter phenomenon was accompanied by evidence of increased vascular permeability in the splanchnic region. The i.v. injection of a bolus of concentrated albumin (25%) also served as an index of the state of the animal. In normal and compensated individuals, such a bolus led to uptake of fluid and restoration of control levels of $\pi p\ell$ within 15-20 minutes. In decompensated animals, such an injection was not associated with fluid uptake and plasma c.o.p. levels remained elevated.

(2) Experiments in which partial resection of the intestines was carried out, it could be shown that hemodilution (as measured by $\pi p\ell$ levels) was suppressed or absent. Hence, the major source of tissue fluid for the vascular system would appear to be the splanchnic viscera, in particular the intestinal tract. Additional attention should be given to fluid replacement therapy by this route.

(3) A membrane osmometer was designed and tested which could be used as a clinical instrument. The unit is small, has a simple temperature control and provides readings within 2-3 minutes for fluid samples as small as 0.5 ml.

(4) The measurement of micropressures and their distribution in the microcirculation during the shock syndrome made it possible to distinguish between vascular and blood derrangements. It was found that microvascular decompensation was the primary defect following hemorrhage, whereas there was some evidence of red blood cell aggregation and poor distribution of blood in the capillary network following lethal doses of bacterial endotoxins.

(5) The ability of the vascular system to maintain capillary pressure (P_c) at a fixed level served as an index of vascular behavior. Periods of hypotension (below 50 mm Hg) in excess of two hours led to a progressive impairment of such vascular regulation. An ominous prognostic feature was the appearance of fluctuations in P_c in an upward and downward erratic course. This type of regulation is believed to represent a myogenic type of response of the smallest precapillary vessels.

(6) In view of the need to balance hydrostatic pressures with plasma colloid osmotic pressures to maintain blood-tissue fluid balance, various volume replacement measures during hemorrhagic shock were evaluated both by following plasma c.o.p. levels and by observing fluid exchange in the microcirculation. A combination of balanced electrolyte infusion followed by the slow administration of 25% albumin was highly effective in restoring both of these features.

(7) Studies on the regional distribution of the circulation after fatal endotoxemia indicated a defective splanchnic adjustment within 90 minutes

after the initial insult.